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Review Article

Overweight, air and noise pollution: Universal risk factors for pediatric pre-hypertension

Roya Kelishadi[1], Parinaz Poursafa[2], Kasra Keramatian[3]

Abstract

Pediatric pre-hypertension (pre-HTN) has a complex multifactorial etiology. Although most cases are secondary to other disorders, a substantial number of children and adolescents have primary or essential HTN and pre-HTN. The gene-gene and gene-environment interactions should be considered in this context. The strong relationship of pre-HTN with environmental factors such as air pollution, noise pollution and passive smoking and obesity suggest that its prevalence will be escalating.

Exposure to ambient particulate matters may increase blood pressure (BP) within hours to days. The underlying biologic pathways include autonomic nervous system imbalance and arterial vascular dysfunction or vasoconstriction because of systemic oxidative stress and inflammation. Likewise, tobacco smoke exposure of pregnant mothers increases systolic BP of their offspring in early infancy. Parental smoking also independently affects systolic BP among healthy pre-school children. Noise exposure, notably in night, is associated with catecholamine secretion, increased BP and a pre-HTN state even in pre-school age children.

Excess weight is associated with dysfunction of the adipose tissue, consisting of enlarged hypertrophied adipocytes, increased infiltration by macrophages and variations in secretion of adipokines and free fatty acids. These changes would result in chronic vascular inflammation, oxidative stress, activation of the renin-angiotensin-aldosterone system and sympathetic response, and ultimately to pre-HTN from childhood.

Prevention and control of the modifiable risk factors of pre-HTN from prenatal period can have long-term health impact on primordial and primary prevention of chronic non-communicable diseases. This review presents a general view on the diagnosis, prevalence and etiology of pre-HTN along with practical measures for its prevention and control.

KEYWORDS: Prevention, Blood Pressure, Pre-hypertension, Genetics, Environment, Children.

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Hypertension (HTN) is the leading risk factor for cardiovascular disease with a worldwide prevalence of near one billion. It is well-established that HTN has a multifactorial etiology; it is a polygenic disease involving a major influence of various environmental factors.[1-5]

A growing body of evidence demonstrated that raised blood pressure (BP) during adulthood has its root in the childhood.[6-8] In other words, high BP during childhood predicts long-term outcome in their future life. Some research on changing cardiovascular markers among hypertensive children as well as autopsy studies demonstrated aortic and heart vessels atherosclerotic changes that support this idea. In addition, it is documented that the higher systolic BP in children predicts the stiffer arteries during adulthood.[7-11]

The supporting data indicates that not only

1- Professor, Department of Paediatrics, Child Health Promotion Research Center, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran
2- Environment Research Center, Isfahan University of Medical Sciences, Isfahan, Iran
3- Child Health Promotion Research Center, Isfahan University of Medical Sciences, Isfahan, Iran
Corresponding Author: Parinaz Poursafa
E-mail: parinaz.poursafa@gmail.com

high BP significantly damages vital organs function in the future life of children and adolescent, but also pre-hypertension (pre-HTN) has the same harmful effects.[9,10,12,13] The Fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents highlighted that all children aged above 3 years who are seen in a medical setting should have their BP measured.[14] It also illustrated the concept of pre-HTN after that the same term was developed for adults by the Joint National Committee on prevention detection evaluation and treatment of high blood pressure seventh report (JNC7). This term is used when a person's BP is elevated above normal but not to the level considered to be HTN.[15]

Given the increasing evidence on tracking of BP from childhood into adult life, the relative contributions of genetic, prenatal, environmental, biological and behavioral determinants to pediatric pre-HTN should be underscored. This review emphasizes on the importance and determinants of pre-HTN among children and adolescents, and highlights the modifiable factors that may be effective in primordial prevention of many chronic non-communicable diseases.

For this review, we identified studies published in the English language from 1990 to 2011, by a World Wide Web-based literature search using PubMed , Medline, Ovid MEDLINE(R) in process and other non-indexed citations, Allied and Complementary Medicine (AMED),Cumulative Index to Nursing and Allied Health Literature (CINAHL) ,Scopus, CAB Abstracts , and Global Health. The following search terms were used: blood pressure, hypertension, hypertensive, high blood pressure, pre-hypertension, lifestyle, environment, genetics, children, adolescents, and prevention. Data on study design and location, confounding factors, health outcomes, and study findings were extracted from the selected studies. We also used secondary references cited by the articles recognized in the primary search.

1. Diagnosis of pre-HTN in children and adolescents

The diagnosis of pre-HTN needs multiple measurements, taken at least in three occasions over a period of time. Home and ambulatory BP measurements are more precise, but are recommended for special cases. In children and adolescents, home BP is lower than daytime ambulatory BP. This difference may be because of the daytime physical activity of this age group. Stage I hypertension is defined as a mean BP level from the 95th percentile to 5mmHg above the 99th percentile. Stage II hypertension is considered as an average BP exceeding 5mmHg above the 99th percentile.[6, 14]

The BP percentiles are constructed in some countries, and have reported slightly higher BP levels in children living in Northern Europe[16,17] and South Asia[18] than those living in the US,[6,14] however a national study in Iran revealed reference curves consistent with the US curves.[19]

During growth, BP increases with age and body size; thus the accurate diagnosis of abnormal BP levels requires the use of standardized charts by age, gender and height.[6,14] Despite the report of Collins et al. which challenged the recent method of diagnosis of these disorders in the pediatric age groups,[20] clinicians use the national high blood pressure education program working group guideline.[6,14,21] In accordance with this guideline, pre-HTN is defined as the average systolic or diastolic blood pressure between 90th and 95th percentile in more than three different visits, and HTN as the average BP values more than 95thpercentile. Quantities lower than 90th percentiles are considerate as the normal BP. The definition of pre-HTN is similar in adolescents of all ages, and is considered as BP > 120/80 mmHg.[6, 14, 21, 22]

The importance of pre-HTN in children and adolescents has different aspects. While increasing evidence is now available on the prevalence of pre-HTN and HTN in the pediatric age group and on escalating number of the cases of essential HTN in adolescence, there is

also evidence of BP tracking, indicating that children and adolescents with pre-HTN tend to maintain that position over time.[6, 22-25]

2. Prevalence of pre-HTN in the pediatric age group

Limited evidences exist on the prevalence of pre-HTN in children and adolescents. A summary of different results is presented in Table 1.[9,10,12,26-40] This prevalence has a wide range, and is usually about 4%, but it has been reported as high as 15.7% in adolescents.[39] Serial BP measurement over time showed that 14% of adolescents with pre-HTN developed HTN in 2 years, which shows an incidence rate of about 7% per year for HTN. In the same study, after two years of follow up, 43% of girls and 68% of boys with high risk BP values developed pre-HTN.[4]

3. Etiology of pre-HTN in children and adolescents

Pediatric HTN and pre-HTN have a complex multifactorial etiology. Although most cases are secondary to causes as renal, cardiovascular or endocrine disorders, a substantial number of children and adolescents are currently diagnosed with primary or essential HTN and pre-HTN.[41] Some of the most important etiologies are as follows:

3.1. Genetic factors

There is a growing body of evidence in detecting mutations and different combinations of genetic variations which may cause HTN and pre-HTN. Potentially variations were identified, which were associated with quantitative differences in the expression of multiple genes such as the differences in expression of the genes coding for the angiotensin-converting enzyme and for the natriuretic peptide receptor.[22,42-48] However the etiology is more complex and the gene-gene and gene-environment interactions should be considered in this context.[49]

3.2. Environmental factors

Exposures to various environmental factors before and after birth have harmful effects on cardiovascular system.[50] Various environmental risk factors are identified for pre-HTN, the most important ones being air pollution, noise pollution and second-hand smoking.

3.2.1. Air pollution and pre-HTN

A growing body of evidence exists about the effect of air pollutants, notably particulate matter (PM) on pre-HTN. This association was found to be independent of aerologic factors like weather, temperature or humidity and of major cardiovascular risk factors such as age, diabetes, dyslipidemia and obesity.[51-54]

Accumulating evidences suggest that exposure to ambient levels of PM may increase BP within hours to days and can result in a prohypertensive response. The underlying biologic pathways include autonomic nervous system imbalance and arterial vascular dysfunction or vasoconstriction because of systemic oxidative stress and inflammation.[22,55,56] Given the harmful effects of air pollutants, notably PM, on various organs and on underlying mechanisms of atherosclerosis and endothelial dysfunction from childhood,[57-64] preventive measures should be considered from early life.
The Multi-Ethnic Study of Atherosclerosis showed that traffic-related exposure may increase systolic BP, and in turn left ventricular mass index. The increase in this index has been consistent with 5.6 mmHg elevation in BP.[65-67] Exposures to air pollutants other than PM, arsenic, lead, cadmium, solvents, and pesticides have also been linked to pre-HTN.[68]

3.2.2. Passive smoking and pediatric pre-HTN

The harmful effects of secondhand smoke on cardiovascular system are comparable to that of smoking.[69] Some studies have documented the association of exposure to tobacco smoke with elevated BP in children and adolescents. Tobacco smoke exposure of pregnant mothers has a considerable effect on increasing systolic BP of their offspring in early infancy.[70]
A recent large population-based study showed that parental smoking independently affects systolic BP among healthy preschool children even after correction for other risk factors, such as body mass index, parental hypertension, or

Table 1. Summary of studies on the prevalence of pediatric pre-hypertension:1990-2011

Reference	Location	Population	Aims	Findings
Stabouli S et al., 2009[9]	Athens, Greece	124 children and adolescents (5 to 18 y)	Investigate the left ventricular mass index (LVMI) and left ventricular hypertrophy (LVH) among pre-hypertensive and hypertensive children.	Hypertensive and pre-hypertensive subjects had significantly higher LVMI as well as more prevalence of LVH than normotensive.
Genovesi S et al., 2008[10]	Milan, Italy	75 children (9.7 ± 0.2 y) subdivided into three groups of normotensive, pre-hypertensive and hypertensive	Assessment of RR intervals and baroreflex impairment	Hypertensive and pre-hypertensive children display a marked baroreflex impairment
Lubrano R et al., 2009[12]	Rome, Italy	cohort study 146 children with pre-HTN 104 normal blood pressure, (9.12 ± 3.28 y)	The effect of Pre-HTN on renal function	Pre-hypertensive group had higher proteinuria and lower GFR
Din-Dzietham R et al., 2007[26]	United states	National survey data from 1963 to 2002. 8- to 17-year-old non-Hispanic blacks and whites and Mexican Americans	The BP, pre-HBP, and HBP trends between 1963 to 2002 The association between BP and sex, ethnic and weight	1) The BP, pre-HTN, and HTN trends were downward from1963 to 1988. 2) Non-Hispanic blacks and Mexican Americans had a greater prevalence of HTN and pre-HTN than non-Hispanic whites 4) Males had a greater prevalence of pre-HTN and HTN than females
Salman Z et al., 2010[27]	Khartoum, Sudan	304 children (6-12 y)	prevalence of HTN and Pre-HTN and its association with obesity	4.9% were pre-hypertensive and 4.9% were hypertensive and its strongly associated with the obesity
Sharma A et al., 2010[28]	northern India in Shimla	1085 students aged 11-17 years	prevalence of HTN and PRE-HTN in comparison with urban and rural and in relation to obesity	1) Pre-HTN in 12.3%. HTN in 5.9% of children. 2) more urban have HTN and more rural children have Pre-HTN 3) HTN and Pre-HTN are more in higher BMI
Ostchega Y et al., 2009 [29]	United states	8-17 years from the National Health and Nutrition Examination Surveys (NHANES)	PRE-HTN and HTN estimation during 2003-2006	13.6% of boys and 5.7% of the girls aged were classified as Pre-HTN. Both obese boys and girls had higher rate of Pre-HTN and HTN.
Hansen ML et al., 2007[30]	Northeast Ohio	14,187 children and adolescents aged 3 to 18 years. cohort study June 1999 until September 2006	determine the frequency of undiagnosed HTN and PRE-HTN	3.4% had pre-HTN vs. 3.6% HTN HTN and pre-HTN were frequently undiagnosed
Salvadori M et al., 2008[31]	Canada	675 children (aged 4-17 years) during the year 2004	Evaluation of the association between overweight and obesity with pre-HTN and HTN in the rural children	Pre-HTN was 7.6% Overweight was associated with HTN but not pre-HTN. Obesity was associated with both of them.

Table 1. Continue

Reference	Location	Population	Aims	Findings
Diaz A et al., 2010[32]	Argentina	331 children(5-11) and adolescents(12-18 y) rural students	prevalence of HTN, sedentary lifestyle, overweight, and obesity	Pre-HTN was detected in 1.9% and 1.7% of children and adolescents.
Aglony IM et al., 2009[33]	Santiago, Chile	112 children (6-12 y)	Assessment of blood pressure, cardiovascular risk factors and family history in healthy children	Pre-HTN was 3.6% and HTN 2.7%. Only one pre-hypertensive child versus all hypertensive ones had family history of HTN
Juarez-Rojas JG et al., 2008[34]	Mexico City	1846 students (12 to 16 years old)	HTN prevalence and cardiovascular risk factors association	Pre-HTN 10% and HTN 10.6%. The higher prevalence of cardiovascular risk factors associated with pre-HTN and HTN
Culhane-Pera KA et al., 2009[35]	Hmong refugees Thailand	988 refugees (0-20 y) June 2004-March 2006	Prevalence of cardiovascular risk factors	Pre-HTN : 9.6% HTN: 8.2%
Liang YJ et al., 2010[36]	China	8247 children and adolescents (6-17 y)	observe the trends in BP during 1991 to 2004	Pre-HTN and HTN increased 6.38% and 8.13% in children and adolescents respectively
Chiolero A et al., 2007[37]	Switzerland	5207 school children (12.3 ± 0.5 y) 2005/2006	Prevalence of pre-HTN and HTN	Pre-HTN:13.3% HTN: 11.4 (first visit) & 2.2% (third visit)
Di Bonito P et al., 2009[38]	Naples, Italy	447 obese and 131 normal-weight children	prevalence of pre-HTN in obese children	Pre-HTN observed in 17.7% of obese children and in 1.5% of controls. Boys were more likely to have pre-HTN.
McNiece KL et al., 2007[39]	Houston, United states	6790 adolescents (11-17 y) 2003 to 2005	prevalence of HTN and pre-HTN	1) At first visit, 9.5% had pre-HTN, and 9.4% HTN. At third visit, 15.7% were pre-hypertensive and 3.2% hypertensive 2) HTN and pre-HTN prevalence were increased in parallel with increasing the BMI
Falkner B et al., 2008[40]	Philadelphia, united states	8533 high school students (13 to 15y)	Evaluate the progression of pre-HTN to HTN	17.22% had pre-HTN.14% of boys and 12% of girls had HTN 2 years later. Overall, 7% per year.

birth weight.[71] Likewise, a family-centered prospective study revealed this association among children and adolescents.[72] This widespread and modifiable risk factor should be considered in primordial and primary prevention of pre-HTN.

3.2.3. Noise pollution and pre-HTN

The cardiovascular effects of environmental noise, notably on high BP, are well documented and rank second in terms of disability-adjusted life year (DALYs) after annoyance.[73-75] It is also documented that environmental noise exposure may be associated with elevated BP in young adults, especially in female individuals.[76]

Night time noise has an effect on our blood pressure more than day time noise. The HYENA (Hypertension and Exposure to Noise near Airports) study was a large study conducted

among individuals who had lived at least 5 years near any of six major European airports. It found significant exposure-response relationships between night-time aircraft and average daily road traffic noise exposure and elevated BP. Monitoring BP showed that systolic BP increased by 6.2 mmHg and diastolic BP by 7.4 mmHg. The association of noise pollution with increased BP remained significant even after adjustment for major confounders as health, socioeconomic and lifestyle factors including diet and physical activity.[77-78]

Noise exposure is associated with increased catecholamine secretion. In children, in addition to impairing reading comprehension and long-term memory, chronic noise exposure may be associated with increased BP and a pre-HTN state.[79-81] Such association is reported even in pre-school age children.[82]

Some changes in BP levels cannot be explained by well-known determinants as anthropometric measures and lifestyle factors. For instance, comparison of data from 4 waves of the Korean National Health and Nutrition Examination Survey between 1998 and 2008 among children and adolescents with 10 to 19 years of age revealed significant decrease in mean BP as well as in the prevalence of pre-HTN and HTN. These changes were not explained by secular trend of childhood obesity, cigarette smoking, physical activity, dietary habits, sociodemographic factors and psychological factors as perceived stress and sleep duration.[83] Such findings may be confirmatory evidence of the underlying role of environmental factors on BP levels and pre-HTN state in children and adolescents.

3.3. Gene-environment interaction and pre-HTN

The interaction of gene and environment on the development of many chronic diseases and their risk factors is well-established. The discrepancies of human genome and modern lifestyle can, at least in part, explain the ongoing epidemics of chronic non-communicable diseases.[84-90]

Likewise, such interaction may have a pivotal role in the development of pre-HTN. A growing body of evidence exists about the link between fetal programming and pre-HTN later in life.[91] The fetal origins of adult disease is related to insults to epigenetic modifications of genes.[92] Such epigenetic process of establishing future diseases may affect some genes responsible for fetal and placental growth.[93]

It is suggested that environmental changes during the prenatal and perinatal periods may be associated with altered gene expression by epigenetic mechanisms resulting in pre-HTN, HTN, and other chronic diseases.[94]

The association of environmental fetal programming is documented in young children; it is shown that in 6-year-old children born at full term, intrauterine growth retardation was linked to pre-HTN.[95] Different mechanisms are considered in this regard, one of them is about the role of oxidative stress. A recent experimental study showed that the intrauterine environment modifies oxidative pathways of gene expression in fetal kidneys, and this may be a mechanism of pre-HTN.[96] It is also suggested that fetal programming of pre-HTN may be mediated by the fundamental role of hyperinsulinism and hyperleptinemia.[97]

Various factors as maternal obesity,[98] dietary habits in pregnancy,[99] and gestational diabetes[100]are considered to be the underlying mechanism of restricted fetal growth, and in turn of pre-HTN. Of special concern in this context is the association of environmental pollutants and other chemical toxins, which may result in intrauterine growth retardation and its sequels. Environmental pollutants may influence vital cellular functions during critical periods of fetal development, and may change the structure or function of vital organs. Developmental epigenetics may lead to "adaptive" phenotypes to respond the needs of the later-life environment. Exposure to environmental pollutants and toxic chemicals may interfere with these programmed adaptive changes, and eventually may result in considerable increase in various disorders as pre-HTN.[101-104]

3.4. Overweight and pediatric pre-HTN

Excess weight is associated with dysfunction of

the adipose tissue, consisting of enlarged hypertrophied adipocytes, increased infiltration by macrophages and variations in secretion of adipokines and free fatty acids. These changes would results in chronic vascular inflammation, oxidative stress, activation of the renin-angiotensin-aldosterone system and sympathetic response, and ultimately to pre-HTN.[105] The association of overweight with pre-HTN and HTN in the pediatric age group is well-documented.[106-114]

A birth cohort demonstrated that maternal pre-pregnancy weight and BMI, and weight at the end of pregnancy are found to be positively associated with both systolic and diastolic BP in adolescent subjects of both sexes; maternal height was positively associated with systolic BP only among males.[115]

The strong association of elevated BP with excess weight along with the childhood obesity epidemic has led to increase in the prevalence of the cases of pediatric HTN. Comparison of national data in the US revealed an increase in mean systolic and diastolic BP levels,[116] and an overall increase in the prevalence of pre-HTN and HTN in children and adolescents.[26,117]

Considering the rapidly escalating trend of childhood obesity, it can be estimated that the population prevalence of pre-HTN and HTN will be increasing from childhood to adulthood. This is of crucial importance for low- and middle-income countries facing increasing levels of childhood obesity[118-122] as well as an emerging epidemic of non-communicable diseases.[123-127]

3.5. Lifestyle factors associated with pediatric pre-HTN

The pre-HTN state during childhood and adolescence is associated with various lifestyle factors. Although smoking may be associated with elevated BP, but such evidence is limited in the pediatric age group. Diet and physical activity are known a pivotal role in the development, prevention and control of pediatric pre-HTN. Their impact may begin from early life.[6, 128]

Various factors have been considered in this regard:

3.5.1. Dietary factors in infancy

Diet is known to be associated with mean BP level, and pre-HTN; this association is documented from early life. Infant-feeding may have lifelong health impact. The protective role of breast feeding against chronic diseases and cardiometabolic risk factors, including elevated BP, is well-established.[129-132] Accumulating evidence exists on the protective effect of breast feeding during infancy on BP and pre-HTN in later life.[19,133]

Results of systematic reviews and meta-analyses confirmed the protective role of breast feeding against elevated BP in later life.[133-135] Although, the correlations were not strong, but even such small reduction in BP associated with breastfeeding could confer important benefits at population level.

A birth cohort showed that after 7.3 years of follow up, children who were bottle-fed during infancy had significantly higher systolic BP than those who were breast-fed.[136] However, some studies have shown small effects of breast feeding on children's BP, and have suggested that such effect may become more evident during adolescence.[137-139]

In addition to the beneficial effects of breast feeding for future BP of infants, it may have protective effects against elevated BP for mothers. A recent study in Finland found that16-20 years after pregnancy, women who had breast-fed for less than 6 months had higher total body fat and cardiometabolic risk factors than mothers who had breast-fed for longer than 6 months or for longer than 10 months. The protective long-term effects of duration of postpartum lactation on risk factors including systolic and diastolic BP were independent of pre-pregnancy weight and BMI, menopausal status, smoking status, level of education, participation in past and present leisure-time physical activity and current dietary energy intake.[140]

The type and the beginning time of complementary foods may have lifelong health impacts.

One of its aspects is the intake of sodium (salt), and related harmful effect on the devel-

oping kidneys and blood pressure. One of the recent evidences in this regard came from a large cohort of 8-month-old infants in the UK. It showed that 70% of infants consumed more than 400 mg sodium per day, which is the maximum UK recommendation for sodium intake in infants.[141]

Given the suggested effect of salt intake from infancy to adulthood on BP, body weight and energy balance,[142] high sodium intake by complimentary foods may increase the cases of pre-HTN in the future.

The important role of taste preference should be taken into account. A recent study showed that healthy and low-sodium foods can be simply introduced in the diet of most infants.[143] Usually the taste preferences in new food acceptance in early life may persist lifelong, and may have long-term health effects. Pediatricians have a pivotal role in introducing healthy complimentary foods to families;[144] and less-educated mothers need to be learned about infant feeding practices.[145]

3.5.2. Diet in childhood and adolescence

Different guidelines have suggested low sodium intake in childhood and adolescence for prevention of pre-HTN and HTN.[146-148] However, the current salt intake is far in excess of nutritional requirements in children and adolescents of many populations.[126,149-153] This high intake is reported even in very young children with 3 years of age.[154]

Of special concern is that the high sodium content is not limited to unhealthy foods as snacks and processed foods, those foods as bread, cheese and cereals considered as healthy with recommendation of daily consumption, are of main hidden sources of salt intake among children and adolescents.[155]

Various types of studies have confirmed the effect of salt intake and pre-HTN in the pediatric age group. A large population-based study in the UK showed that the increase in salt intake by 1g in children is associated with 0.4 mmHg rise in systolic BP and 0.6 mmHg rise in pulse pressure.[156]

It is also documented that in addition to the direct association of salt intake on BP, excess dietary sodium intake would affect thirst and may increase consumption of sugary beverages by children and adolescents; and in turn, it would increase the likelihood of obesity and pre-HTN.[157]

A meta-analysis of controlled trials assessed the effect of reducing salt intake on BP in children and adolescents demonstrated that a modest reduction in salt intake resulted in immediate decline in BP level.[158]

However, the effects of low-sodium intake on pre-HTN are controversial, and may be not generalizable. A Cochrane review suggested that the effect of low versus high sodium intake on blood pressure was greater in Black and Asian patients than in Caucasians. Magnitude of the effect in Caucasians with normal blood pressure does not warrant a general recommendation to reduce sodium intake.[159]

On the other hand, some harmful effects of lowering salt intake are reported. In a recent study among healthy participants, low-salt diet was independently associated with an increase in insulin resistance.[160]

The dietary recommendations for prevention and control of pediatric pre-HTN are not limited to reducing the salt intake. Findings of the 8-year follow up of children initially 3 to 6 years of age in the prospective Framingham Children's Study showed children who consumed more fruits and vegetables or more dairy products during the preschool age had smaller yearly increase in systolic BP in subsequent years. At early adolescence, children with higher intakes of fruits and vegetables and dairy products had a lower mean systolic BP than those with either high intake of one of these food groups or those with low intake of them. This longitudinal study confirmed that a diet rich in fruits, vegetables, and dairy products may have beneficial effects on BP during childhood.[161]This type of diet is consistent with the classic Dietary Approaches to Stop Hypertension (DASH) including a diet high in fresh fruits, vegetables, whole grains, and low-fat dairy products, recommended for adults.[162]

The beneficial effects of a DASH-type diet on cardiometabolic risk factors of children and adolescents were documented too. A large national study in the US showed that after 10 years of follow up, adolescent girls with diet more close to the DASH eating pattern had smaller gains in BMI.[163] Trials with dietary interventions using the DASH-type diet showed favorable results in controlling pre-HTN state in diabetic[164,165] and non-diabetic adolescents.[166] These findings highlight the need to underscore the intake of fruits, vegetables, fiber and dairy in the diets of children and adolescents.

3.5.3. Physical activity and pre-HTN

Physical activity is known to positively moderate BP. There is a large body of evidence about the association of physical activity and lower cardiovascular risk factors, including pre-HTN and HTN.[9, 32,113,167,168] A study among young children showed that physical activity was independently associated with lower BP even in prepubertal children.[169]

A recent systematic review confirmed the association of physical activity with lower BP in children, and emphasized on the role of physical fitness on prevention and control of cardiovascular risk factors in children.[170]

Although it is well documented that sedentary behaviors are associated with pre-HTN, but a recent study among adolescents demonstrated various correlations between different types of sedentary activities and BP levels. It showed that after adjusting for confounders, while each hour per day spent in screen time, watching TV and playing video games was associated with a significant increase in diastolic BP, each hour per day spent reading was associated with a decrease in systolic and diastolic BP.[171]

Another national study in the US revealed that high TV use, but not high computer use, and lack of moderate-to-vigorous intensity physical activity was associated with cardiometabolic risk factors among children and adolescents.[172] Office- and population-based interventions have demonstrated that pre-HTN may be reversible by increase in physical activity.[173-176]

Among different types of physical activities, aerobic exercises are found to be useful for controlling pre-HTN.[177,178] Likewise , this kind of exercise has been successful in reducing systolic and diastolic BP among children and adolescents with pre-HTN and HTN.[179]

In general, a combination of lifestyle change including healthy dietary pattern and exercise training may improve pre-HTN state and vascular function in children and adolescents.[6,180,181]

Conclusion

Childhood onset of adult non-communicable diseases has become a substantial health problem. The strong relationship of pre-HTN with environmental factors and obesity along with the increasing environmental pollution and the childhood obesity epidemic suggest that the population prevalence of pediatric pre-HTN will be escalating, and there is already evidence that this trend is proceeding. Physicians and health professionals who care for children and adolescents should incorporate screening of adult non-communicable diseases into their practice. Prevention and control of pediatric pre-HTN is of crucial importance in primary prevention of such chronic diseases. Prevention and control of its modifiable risk factors such as air and noise pollution, passive smoking, overweight and unhealthy lifestyle, along with primordial prevention by good pregnancy care for prevention of low birth weight, encouraging breast feeding, and using healthy complimentary foods during infancy can impact the overall health of children and adolescents, as well as the prevention of chronic non-communicable diseases. Better knowledge on the etiology of these disease states will help preventive and targeted therapies.

Conflict of Interests
Authors have no conflict of interests.

Authors' Contributions
RK planned and conducted the review, wrote and finalized it, PP and KK assisted in planning, conducting and writing the review. All authors read and approved the final draft of the paper.

References

1. Olafiranye O, Zizi F, Brimah P, Jean-Louis G, Makaryus AN, McFarlane S, et al. Management of Hypertension among Patients with Coronary Heart Disease. Int J Hypertens 2011; 2011: 653903.
2. Clara JG, De Macedo ME, Pego M. Prevalence of isolated systolic hypertension in the population over 55 years old. Results from a national study. Rev Port Cardiol 2007; 26(1): 11-8.
3. Kearney PM, Whelton M, Reynolds K, Whelton PK, He J. Worldwide prevalence of hypertension: a systematic review. J Hypertens 2004; 22(1): 11-9.
4. Rosamond W, Flegal K, Friday G, Furie K, Go A, Greenlund K, et al. Heart disease and stroke statistics--2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2007; 115(5): e69-171.
5. Mittal BV, Singh AK. Hypertension in the developing world: challenges and opportunities. Am J Kidney Dis 2010; 55(3): 590-8.
6. Balagopal PB, de Ferranti SD, Cook S, Daniels SR, Gidding SS, Hayman LL, et al. Nontraditional risk factors and biomarkers for cardiovascular disease: mechanistic, research, and clinical considerations for youth: a scientific statement from the American Heart Association. Circulation 2011; 123(23): 2749-69.
7. Sun SS, Grave GD, Siervogel RM, Pickoff AA, Arslanian SS, Daniels SR. Systolic blood pressure in childhood predicts hypertension and metabolic syndrome later in life. Pediatrics 2007; 119(2): 237-46.
8. Berenson GS, Srinivasan SR, Bao W, Newman WP, III, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med 1998; 338(23): 1650-6.
9. Stabouli S, Kotsis V, Rizos Z, Toumanidis S, Karagianni C, Constantopoulos A, et al. Left ventricular mass in normotensive, prehypertensive and hypertensive children and adolescents. Pediatr Nephrol 2009; 24(8): 1545-51.
10. Genovesi S, Pieruzzi F, Giussani M, Tono V, Stella A, Porta A, et al. Analysis of heart period and arterial pressure variability in childhood hypertension: key role of baroreflex impairment. Hypertension 2008; 51(5): 1289-94.
11. Li S, Chen W, Srinivasan SR, Berenson GS. Childhood blood pressure as a predictor of arterial stiffness in young adults: the bogalusa heart study. Hypertension 2004; 43(3): 541-6.
12. Lubrano R, Travasso E, Raggi C, Guido G, Masciangelo R, Elli M. Blood pressure load, proteinuria and renal function in pre-hypertensive children. Pediatr Nephrol 2009; 24(4): 823-31.
13. Lucini D, Mela GS, Malliani A, Pagani M. Impairment in cardiac autonomic regulation preceding arterial hypertension in humans: insights from spectral analysis of beat-by-beat cardiovascular variability. Circulation 2002; 106(21): 2673-9.
14. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004; 114(2): 555-76.
15. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jr., et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003; 42(6): 1206-52.
16. Jackson LV, Thalange NK, Cole TJ. Blood pressure centiles for Great Britain. Arch Dis Child 2007; 92(4): 298-303.
17. Munkhaugen J, Lydersen S, Wideroe TE, Hallan S. Blood pressure reference values in adolescents: methodological aspects and suggestions for Northern Europe tables based on the Nord-Trondelag Health Study II. J Hypertens 2008; 26(10): 1912-8.
18. Sung RY, Choi KC, So HK, Nelson EA, Li AM, Kwok CW, et al. Oscillometrically measured blood pressure in Hong Kong Chinese children and associations with anthropometric parameters. J Hypertens 2008; 26(4): 678-84.
19. Kelishadi R, Ardalan G, Gheiratmand R, Majdzadeh R, Delavari A, Heshmat R, et al. Blood pressure and its influencing factors in a national representative sample of Iranian children and adolescents: the CASPIAN Study. Eur J Cardiovasc Prev Rehabil 2006; 13(6): 956-63.
20. Collins RT, Alpert BS. Pre-hypertension and hypertension in pediatrics: don't let the statistics hide the pathology. J Pediatr 2009; 155(2): 165-9.

21. Update on the 1987 Task Force Report on High Blood Pressure in Children and Adolescents: a working group report from the National High Blood Pressure Education Program. National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. Pediatrics 1996; 98(4 Pt 1): 649-58.
22. Long AN, Dagogo-Jack S. Comorbidities of diabetes and hypertension: mechanisms and approach to target organ protection. J Clin Hypertens (Greenwich) 2011; 13(4): 244-51.
23. Gidding SS. Measuring children's blood pressure matters. Circulation 2008; 117(25): 3163-4.
24. Bao W, Threefoot SA, Srinivasan SR, Berenson GS. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: the Bogalusa Heart Study. Am J Hypertens 1995; 8(7): 657-65.
25. Fuentes RM, Notkola IL, Shemeikka S, Tuomilehto J, Nissinen A. Tracking of systolic blood pressure during childhood: a 15-year follow-up population-based family study in eastern Finland. J Hypertens 2002; 20(2): 195-202.
26. Din-Dzietham R, Liu Y, Bielo MV, Shamsa F. High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. Circulation 2007; 116(13): 1488-96.
27. Salman Z, Kirk GD, Deboer MD. High Rate of Obesity-Associated Hypertension among Primary Schoolchildren in Sudan. Int J Hypertens 2010; 2011: 629492.
28. Sharma A, Grover N, Kaushik S, Bhardwaj R, Sankhyan N. Prevalence of hypertension among schoolchildren in Shimla. Indian Pediatr 2010; 47(10): 873-6.
29. Ostchega Y, Carroll M, Prineas RJ, McDowell MA, Louis T, Tilert T. Trends of elevated blood pressure among children and adolescents: data from the National Health and Nutrition Examination Survey1988-2006. Am J Hypertens 2009; 22(1): 59-67.
30. Hansen ML, Gunn PW, Kaelber DC. Underdiagnosis of hypertension in children and adolescents. JAMA 2007; 298(8): 874-9.
31. Salvadori M, Sontrop JM, Garg AX, Truong J, Suri RS, Mahmud FH, et al. Elevated blood pressure in relation to overweight and obesity among children in a rural Canadian community. Pediatrics 2008; 122(4): e821-e827.
32. Diaz A, Tringler M, Molina JD, Diaz MC, Geronimi V, Aguera D, et al. [Blood pressure control and arterial hypertension in children and adolescents from a rural population in Argentina: preliminary data from Vela Proyect]. Arch Argent Pediatr 2010; 108(1): 68-70.
33. Aglony IM, Arnaiz GP, Acevedo BM, Barja YS, Marquez US, Guzman AB, et al. [Blood pressure and family history of hypertension in children from Santiago, Chile]. Rev Med Chil 2009; 137(1): 39-45.
34. Juarez-Rojas JG, Cardoso-Saldana GC, Posadas-Sanchez R, Medina-Urrutia AX, Yamamoto-Kimura L, Posadas-Romero C. Blood pressure and associated cardiovascular risk factors in adolescents of Mexico City. Arch Cardiol Mex 2008; 78(4): 384-91.
35. Culhane-Pera KA, Moua M, DeFor TA, Desai J. Cardiovascular disease risks in Hmong refugees from Wat Tham Krabok, Thailand. J Immigr Minor Health 2009; 11(5): 372-9.
36. Liang YJ, Xi B, Hu YH, Wang C, Liu JT, Yan YK, et al. Trends in blood pressure and hypertension among Chinese children and adolescents: China Health and Nutrition Surveys 1991-2004. Blood Press 2010; 20(1): 45-53.
37. Chiolero A, Paccaud F, Bovet P. Pre-hypertension and hypertension among adolescents of Switzerland. J Pediatr 2007; 151(6): e24-e25.
38. Di BP, Forziato C, Sanguigno E, Di FT, Saitta F, Iardino MR, et al. Prehypertension in outpatient obese children. Am J Hypertens 2009; 22(12): 1309-13.
39. McNiece KL, Poffenbarger TS, Turner JL, Franco KD, Sorof JM, Portman RJ. Prevalence of hypertension and pre-hypertension among adolescents. J Pediatr 2007; 150(6): 640-4, 644.
40. Falkner B, Gidding SS, Portman R, Rosner B. Blood pressure variability and classification of prehypertension and hypertension in adolescence. Pediatrics 2008; 122(2): 238-42.
41. Sanjad SA. Etiology of hypertension in children and adolescents. J Med Liban 2010; 58(3): 142-5.
42. Smithies O, Kim HS, Takahashi N, Edgell MH. Importance of quantitative genetic variations in the etiology of hypertension. Kidney Int 2000; 58(6): 2265-80.
43. Melo LG, Steinhelper ME, Pang SC, Tse Y, Ackermann U. ANP in regulation of arterial pressure and fluid-electrolyte balance: lessons from genetic mouse models. Physiol Genomics 2000; 3(1): 45-58.
44. Kardia SL. Context-dependent genetic effects in hypertension. Curr Hypertens Rep 2000; 2(1): 32-8.
45. Tan KT, Dempsey A, Liew CC. Cardiac genes and gene databases for cardiovascular disease genetics. Curr Hypertens Rep 1999; 1(1): 51-8.
46. Paul M. [The renin-angiotensin system as the basic principle for hypertension and coronary heart diseases--role of genetic factors]. Z Kardiol 2000; 89(4): 264-8.
47. Marcano AC, Onipinla AK, Caulfield MJ, Munroe PB. Recent advances in the identification of genes for human hypertension. Expert Rev Cardiovasc Ther 2005; 3(4): 733-41.
48. Dorn GW. The genomic architecture of sporadic heart failure. Circ Res 2011; 108(10): 1270-83.

49. Moore JH, Williams SM. New strategies for identifying gene-gene interactions in hypertension. Ann Med 2002; 34(2): 88-95.
50. Mone SM, Gillman MW, Miller TL, Herman EH, Lipshultz SE. Effects of environmental exposures on the cardiovascular system: prenatal period through adolescence. Pediatrics 2004; 113(4 Suppl): 1058-69.
51. Roman AO, Prieto CM, Mancilla FP, Astudillo OP, Dussaubat AA, Miguel WC, et al. [Association between air pollution and cardiovascular risk]. Rev Med Chil 2009; 137(9): 1217-24.
52. Walker B, Jr., Mouton CP. Environmental influences on cardiovascular health. J Natl Med Assoc 2008; 100(1): 98-102.
53. Brook RD. Is air pollution a cause of cardiovascular disease? Updated review and controversies. Rev Environ Health 2007; 22(2): 115-37.
54. Poursafa P KR. What health professionals should know about the health effects of air pollution and climate change on children and pregnant mothers. Iran J Nurse Midwifery Res 2011; 16(3): 1-8.
55. Brook RD, Rajagopalan S, Pope CA, III, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation 2010; 121(21): 2331-78.
56. Johnson D, Parker JD. Air pollution exposure and self-reported cardiovascular disease. Environ Res 2009; 109(5): 582-9.
57. Kelishadi R, Mirghaffari N, Poursafa P, Gidding SS. Lifestyle and environmental factors associated with inflammation, oxidative stress and insulin resistance in children. Atherosclerosis 2009; 203(1): 311-9.
58. Poursafa P, Kelishadi R, Lahijanzadeh A, Modaresi M, Javanmard SH, Assari R, et al. The relationship of air pollution and surrogate markers of endothelial dysfunction in a population-based sample of children. BMC Public Health 2011; 11: 115.
59. Poursafa P, Kelishadi R, Moattar F, Rafiee L, Amin MM, Lahijanzadeh A, et al. Genetic variation in the association of air pollutants with a biomarker of vascular injury in children and adolescents in Isfahan, Iran. J Res Med Sci 2011; 16(6): 733-40.
60. KelishadiR PP. Air pollution and non-respiratory health hazards for children. Arch Med Sci 2010; 6(4): 483-95.
61. Poursafa P, Kelishadi R, Amini A, Amini A, Amin MM, Lahijanzadeh M, et al. Association of air pollution and hematologic parameters in children and adolescents. J Pediatr (Rio J) 2011; 87(4): 350-6.
62. Kargarfard M, Poursafa P, Rezanejad S, Mousavinasab F. Effects of exercise in polluted air on the aerobic power, serum lactate level and cell blood count of active individuals. Int J Prev Med 2011; 2(3): 145-50.
63. Mansourian M, Javanmard SH, Poursafa P, Kelishadi R. Air pollution and hospitalization for respiratory diseases among children in isfahan, iran. Ghana Med J 2010; 44(4): 138-43.
64. Poursafa P, Kelishadi R. Air pollution, platelet activation and atherosclerosis. Inflamm Allergy Drug Targets 2010; 9(5): 387-92.
65. Van Hee VC, Adar SD, Szpiro AA, Barr RG, Roux AD, Bluemke DA, et al. Common genetic variation, residential proximity to traffic exposure, and left ventricular mass: the multi-ethnic study of atherosclerosis. Environ Health Perspect 2010; 118(7): 962-9.
66. Van Hee VC, Adar SD, Szpiro AA, Barr RG, Bluemke DA, Diez Roux AV, et al. Exposure to traffic and left ventricular mass and function: the Multi-Ethnic Study of Atherosclerosis. Am J Respir Crit Care Med 2009; 179(9): 827-34.
67. Auchincloss AH, Diez Roux AV, Dvonch JT, Brown PL, Barr RG, Daviglus ML, et al. Associations between recent exposure to ambient fine particulate matter and blood pressure in the Multi-ethnic Study of Atherosclerosis (MESA). Environ Health Perspect 2008; 116(4): 486-91.
68. Bhatnagar A. Environmental cardiology: studying mechanistic links between pollution and heart disease. Circ Res 2006; 99(7): 692-705.
69. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. Circulation 2005; 111(20): 2684-98.
70. Geerts CC, Grobbee DE, van der Ent CK, de Jong BM, van der Zalm MM, van Putte-Katier N, et al. Tobacco smoke exposure of pregnant mothers and blood pressure in their newborns: results from the wheezing illnesses study Leidsche Rijn birth cohort. Hypertension 2007; 50(3): 572-8.
71. Simonetti GD, Schwertz R, Klett M, Hoffmann GF, Schaefer F, Wuhl E. Determinants of blood pressure in preschool children: the role of parental smoking. Circulation 2011; 123(3): 292-8.
72. Schwandt P, Haas GM, Liepold E. Lifestyle and cardiovascular risk factors in 2001 child-parent pairs: the PEP Family Heart Study. Atherosclerosis 2010; 213(2): 642-8.
73. Lercher P, Botteldooren D, Widmann U, Uhrner U, KammeringerE.Cardiovascular effects of environmental noise: research in Austria.Noise Health 2011; 13(52): 234-50.

74. Lercher P, Botteldooren D, Widmann U, Uhrner U, Kammeringer E. Cardiovascular effects of environmental noise: research in Austria. Noise Health 2011; 13(52): 234-50.
75. Vangelova KK, Deyanov CE. Blood pressure and serum lipids in industrial workers under intense noise and a hot environment. Rev Environ Health 2007; 22(4): 303-11.
76. Belojevic G, Paunovic K, Jakovljevic B, Stojanov V, Ilic J, Slepcevic V, et al. Cardiovascular effects of environmental noise: research in Serbia. Noise Health 2011; 13(52): 217-20.
77. Chang TY, Lai YA, Hsieh HH, Lai JS, Liu CS. Effects of environmental noise exposure on ambulatory blood pressure in young adults. Environ Res 2009; 109(7): 900-5.
78. Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, et al. Hypertension and exposure to noise near airports: the HYENA study. Environ Health Perspect 2008; 116(3): 329-33.
79. Jarup L, Dudley ML, Babisch W, Houthuijs D, Swart W, Pershagen G, et al. Hypertension and Exposure to Noise near Airports (HYENA): study design and noise exposure assessment. Environ Health Perspect 2005; 113(11): 1473-8.
80. Stansfeld SA, Matheson MP. Noise pollution: non-auditory effects on health. Br Med Bull 2003; 68: 243-57.
81. Cohen S, Evans GW, Krantz DS, Stokols D. Physiological, motivational, and cognitive effects of aircraft noise on children: moving from the laboratory to the field. Am Psychol 1980; 35(3): 231-43.
82. Cohen S, Krantz DS, Evans GW, Stokols D, Kelly S. Aircraft noise and children: Longitudinal and cross-sectional evidence on adaptation to noise and the effectiveness of noise abatement. J Pers Soc Psychol 1981; 40(2): 331-45.
83. Regecova V, Kellerova E. Effects of urban noise pollution on blood pressure and heart rate in preschool children. J Hypertens 1995; 13(4): 405-12.
84. Khang YH, Lynch JW. Exploring determinants of secular decreases in childhood blood pressure and hypertension. Circulation 2011; 124(4): 397-405.
85. Redon J, Cifkova R, Laurent S, Nilsson P, Narkiewicz K, Erdine S, et al. The metabolic syndrome in hypertension: European society of hypertension position statement. J Hypertens 2008; 26(10): 1891-900.
86. Eaton SB, Eaton SB. An evolutionary perspective on human physical activity: implications for health. Comp Biochem Physiol A Mol Integr Physiol 2003; 136(1): 153-9.
87. Rosenberg A. The IUGR newborn. Semin Perinatol 2008; 32(3): 219-24.
88. Guan J, Mao C, Feng X, Zhang H, Xu F, Geng C, et al. Fetal development of regulatory mechanisms for body fluid homeostasis. Braz J Med Biol Res 2008; 41(6): 446-54.
89. Nomura Y, Brooks-Gunn J, Davey C, Ham J, Fifer WP. The role of perinatal problems in risk of co-morbid psychiatric and medical disorders in adulthood. Psychol Med 2007; 37(9): 1323-34.
90. Picone O, Servely JL, Chavatte-Palmer P. [Developmental origin of human adult disease: which importance for obstetrical practice?]. J Gynecol Obstet Biol Reprod (Paris) 2007; 36(4): 338-43.
91. Haimov-Kochman R. [Fetal programming--the intrauterine origin of adult morbidity]. Harefuah 2005; 144(2): 97-101, 151, 150.
92. Redon J, Cifkova R, Laurent S, Nilsson P, Narkiewicz K, Erdine S, et al. Mechanisms of hypertension in the cardiometabolic syndrome. J Hypertens 2009; 27(3): 441-51.
93. Xu XF, Du LZ. Epigenetics in neonatal diseases. Chin Med J (Engl) 2010; 123(20): 2948-54.
94. Reik W, Dean W, Walter J. Epigenetic reprogramming in mammalian development. Science 2001; 293(5532): 1089-93.
95. Waterland RA, Jirtle RL. Early nutrition, epigenetic changes at transposons and imprinted genes, and enhanced susceptibility to adult chronic diseases. Nutrition 2004; 20(1): 63-8.
96. Shankaran S, Das A, Bauer CR, Bada H, Lester B, Wright L, et al. Fetal origin of childhood disease: intrauterine growth restriction in term infants and risk for hypertension at 6 years of age. Arch Pediatr Adolesc Med 2006; 160(9): 977-81.
97. Ghulmiyyah LM, Costantine MM, Yin H, Tamayo E, Clark SM, Hankins GD, et al. The role of oxidative stress in the developmental origin of adult hypertension. Am J Obstet Gynecol 2011.
98. Vickers MH, Breier BH, Cutfield WS, Hofman PL, Gluckman PD. Fetal origins of hyperphagia, obesity, and hypertension and postnatal amplification by hypercaloric nutrition. Am J Physiol Endocrinol Metab 2000; 279(1): E83-E87.
99. Ornoy A. Prenatal origin of obesity and their complications: Gestational diabetes, maternal overweight and the paradoxical effects of fetal growth restriction and macrosomia. Reprod Toxicol 2011; 32(2): 205-12.
100. Symonds ME SSHMBH. Nutritional programming of the metabolic syndrome. Nat Rev Endocrinol 2009; 5(11): 604-11.
101. Nehiri T, Duong Van Huyen JP, Viltard M, Fassot C, Heudes D, Freund N, et al. Exposure to maternal diabetes induces salt-sensitive hypertension and impairs renal function in adult rat offspring. Diabetes 2008; 57(8): 2167-75.

102. Bezek S, Ujhazy E, Mach M, Navarova J, Dubovicky M. Developmental origin of chronic diseases: toxicological implication. Interdiscip Toxicol 2008; 1(1): 29-31.
103. Wen X, Triche EW, Hogan JW, Shenassa ED, Buka SL. Prenatal factors for childhood blood pressure mediated by intrauterine and/or childhood growth? Pediatrics 2011; 127(3): e713-e721.
104. Dotsch J, Plank C, Amann K. Fetal programming of renal function. Pediatr Nephrol 2011.
105. Dores H, Santos P, Salvador F, Maia J, Paixao L, Pereira R, et al. Blood pressure in young adults. Rev Port Cardiol 2010; 29(10): 1495-508.
106. Dorresteijn JA, Visseren FL, Spiering W. Mechanisms linking obesity to hypertension. Obes Rev 2011.
107. Flynn JT, Falkner BE. Obesity hypertension in adolescents: epidemiology, evaluation, and management. J Clin Hypertens (Greenwich) 2011; 13(5): 323-31.
108. de Almeida FA, Konigsfeld HP, Machado LM, Canadas AF, Issa EY, Giordano RH, et al. Assessment of social and economic influences on blood pressure of adolescents in public and private schools: an epidemiological study. J Bras Nefrol 2011; 33(2): 142-9.
109. Knight JA. Diseases and disorders associated with excess body weight. Ann Clin Lab Sci 2011; 41(2): 107-21.
110. Sangun O, Dundar B, Kosker M, Pirgon O, Dundar N. Prevalence of Metabolic Syndrome in Obese Children and Adolescents using Three Different Criteria and Evaluation of Risk Factors. J Clin Res Pediatr Endocrinol 2011; 3(2): 70-6.
111. Saab PG, Fitzpatrick S, Lai B, McCalla JR. Elevated body mass index and obesity among ethnically diverse adolescents. Ethn Dis 2011; 21(2): 176-82.
112. Zhang YX, Wang SR. The relationship of body mass index distribution to relatively high blood pressure among children and adolescents in Shandong, China. Ann Hum Biol 2011; 38(5): 630-4.
113. Zhang YX, Wang SR. Monitoring of blood pressure in overweight and obese children in Shandong, China. Ann Hum Biol 2011; 38(5): 603-7.
114. Leung LC, Sung RY, So HK, Wong SN, Lee KW, Lee KP, et al. Prevalence and risk factors for hypertension in Hong Kong Chinese adolescents: waist circumference predicts hypertension, exercise decreases risk. Arch Dis Child 2011; 96(9): 804-9.
115. Yeste D, Carrascosa A. [Obesity-related metabolic disorders in childhood and adolescence]. An Pediatr (Barc) 2011; 75(2): 135-9.
116. Laura HC, Menezes AB, Noal RB, Hallal PC, Araujo CL. Maternal anthropometric characteristics in pregnancy and blood pressure among adolescents: 1993 live birth cohort, Pelotas, southern Brazil. BMC Public Health 2010; 10: 434.
117. Wang Y, Zhang Q. Are American children and adolescents of low socioeconomic status at increased risk of obesity? Changes in the association between overweight and family income between 1971 and 2002. Am J ClinNutr. 2006; 84(4): 707-16.
118. Martorell R, Kettel KL, Hughes ML, Grummer-Strawn LM. Overweight and obesity in preschool children from developing countries. Int J Obes Relat Metab Disord 2000; 24(8): 959-67.
119. Low LC. Childhood obesity in developing countries. World J Pediatr 2010; 6(3): 197-9.
120. Kelishadi R. Childhood overweight, obesity, and the metabolic syndrome in developing countries. Epidemiol Rev 2007; 29: 62-76.
121. Gupta DK, Shah P, Misra A, Bharadwaj S, Gulati S, Gupta N, et al. Secular trends in prevalence of overweight and obesity from 2006 to 2009 in urban asian Indian adolescents aged 14-17 years. PLoS One 2011; 6(2): e17221.
122. de OM, Blossner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. Am J Clin Nutr 2010; 92(5): 1257-64.
123. Dans A, Ng N, Varghese C, Tai ES, Firestone R, Bonita R. The rise of chronic non-communicable diseases in southeast Asia: time for action. Lancet 2011; 377(9766): 680-9.
124. Bovet P, Viswanathan B, Shamlaye C, Romain S, Gedeon J. Addressing non-communicable diseases in the Seychelles: towards a comprehensive plan of action. Glob Health Promot 2010; 17(2 Suppl): 37-40.
125. Dalal S, Beunza JJ, Volmink J, Adebamowo C, Bajunirwe F, Njelekela M, et al. Non-communicable diseases in sub-Saharan Africa: what we know now. Int J Epidemiol 2011; 40(4): 885-901.
126. Misra A, Singhal N, Sivakumar B, Bhagat N, Jaiswal A, Khurana L. Nutrition Transition in India: Secular Trends in Dietary Intake and their Relationship to Diet-related Non-communicable Diseases. J Diabetes 2011.
127. Al-Daghri NM, Al-Attas OS, Alokail MS, Alkharfy KM, Yousef M, Sabico SL, et al. Diabetes mellitus type 2 and other chronic non-communicable diseases in the central region, Saudi Arabia (riyadh cohort 2): a decade of an epidemic. BMC Med 2011; 9: 76.
128. Katona E, Zrinyi M, Komonyi E, Lengyel S, Paragh G, Zatik J, et al. Factors influencing adolescent blood pressure: the Debrecen Hypertension Study. Kidney Blood Press Res 2011; 34(3): 188-95.

129. de Armas MG, Megias SM, Modino SC, Bolanos PI, Guardiola PD, Alvarez TM. [Importance of breastfeeding in the prevalence of metabolic syndrome and degree of childhood obesity]. Endocrinol Nutr 2009; 56(8): 400-3.
130. Plagemann A, Harder T. Breast feeding and the risk of obesity and related metabolic diseases in the child. Metab Syndr Relat Disord 2005; 3(3): 222-32.
131. Turck D. Later effects of breastfeeding practice: the evidence. Nestle Nutr Workshop Ser Pediatr Program 2007; 60: 31-9.
132. Schack-Nielsen L, Michaelsen KF. Advances in our understanding of the biology of human milk and its effects on the offspring. J Nutr 2007; 137(2): 503S-10S.
133. Smithers L, McIntyre E. The impact of breastfeeding--translating recent evidence for practice. Aust Fam Physician 2010; 39(10): 757-60.
134. Martin RM, Gunnell D, Smith GD. Breastfeeding in infancy and blood pressure in later life: systematic review and meta-analysis. Am J Epidemiol 2005; 161(1): 15-26.
135. Owen CG, Whincup PH, Gilg JA, Cook DG. Effect of breast feeding in infancy on blood pressure in later life: systematic review and meta-analysis. BMJ 2003; 327(7425): 1189-95.
136. Wilson AC, Forsyth JS, Greene SA, Irvine L, Hau C, Howie PW. Relation of infant diet to childhood health: seven year follow up of cohort of children in Dundee infant feeding study. BMJ 1998; 316(7124): 21-5.
137. de Jonge LL, van Osch-Gevers L, Geelhoed JJ, Hofman A, Steegers EA, Helbing WA, et al. Breastfeeding is not associated with left cardiac structures and blood pressure during the first two years of life. The Generation R Study. Early Hum Dev 2010; 86(8): 463-8.
138. Fall CH, Borja JB, Osmond C, Richter L, Bhargava SK, Martorell R, et al. Infant-feeding patterns and cardiovascular risk factors in young adulthood: data from five cohorts in low- and middle-income countries. Int J Epidemiol 2011; 40(1): 47-62.
139. Fewtrell MS. Breast-feeding and later risk of CVD and obesity: evidence from randomised trials. Proc Nutr Soc 2011; 1-6.
140. Wiklund P, Xu L, Lyytikainen A, Saltevo J, Wang Q, Volgyi E, et al. Prolonged breast-feeding protects mothers from later-life obesity and related cardio-metabolic disorders. Public Health Nutr 2011; 1-8.
141. Cribb VL, Warren JM, Emmett PM. Contribution of inappropriate complementary foods to the salt intake of 8-month-old infants. Eur J Clin Nutr 2011.
142. Coelho MS, Passadore MD, Gasparetti AL, Bibancos T, Prada PO, Furukawa LL, et al. High- or low-salt diet from weaning to adulthood: effect on body weight, food intake and energy balance in rats. Nutr Metab Cardiovasc Dis 2006; 16(2): 148-55.
143. Schwartz C, Chabanet C, Lange C, Issanchou S, Nicklaus S. The role of taste in food acceptance at the beginning of complementary feeding. Physiol Behav 2011; 104(4): 646-52.
144. Savino F, Zannino L, Laccisaglia A, Maccario S, Cresi F, Silvestro L, et al. Infant nutritional recommendations from pediatricians. Epidemiologic survey of feeding recommendations for the first year of life in Piedmont. Minerva Pediatr 2004; 56(1): 73-82.
145. Fein SB, Labiner-Wolfe J, Scanlon KS, Grummer-Strawn LM. Selected complementary feeding practices and their association with maternal education. Pediatrics 2008; 122 Suppl 2: S91-S97.
146. Kavey RE, Allada V, Daniels SR, Hayman LL, McCrindle BW, Newburger JW, et al. Cardiovascular risk reduction in high-risk pediatric patients: a scientific statement from the American Heart Association Expert Panel on Population and Prevention Science; the Councils on Cardiovascular Disease in the Young, Epidemiology and Prevention, Nutrition, Physical Activity and Metabolism, High Blood Pressure Research, Cardiovascular Nursing, and the Kidney in Heart Disease; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research. J Cardiovasc Nurs 2007; 22(3): 218-53.
147. Kones R. Is prevention a fantasy, or the future of medicine? A panoramic view of recent data, status, and direction in cardiovascular prevention. Ther Adv Cardiovasc Dis 2011; 5(1): 61-81.
148. Gidding SS, Lichtenstein AH, Faith MS, Karpyn A, Mennella JA, Popkin B, et al. Implementing American Heart Association pediatric and adult nutrition guidelines: a scientific statement from the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. Circulation 2009; 119(8): 1161-75.
149. Hilbig A, Kersting M. 24h-Sodium excretion andhydration status in children and adolescents - Results of the DONALD Study. ClinNutr. 2011. [Epub ahead of print]
150. Eloranta AM, Lindi V, Schwab U, Kiiskinen S, Kalinkin M, Lakka HM, et al. Dietary factors and their associations with socioeconomic background in Finnish girls and boys 6-8 years of age: the PANIC Study. Eur J Clin Nutr 2011.

151. Kawano Y. Salt intake in children. Hypertens Res 2011; 34(7): 797-8.
152. Girardet JP, Rieu D, Bocquet A, Bresson JL, Chouraqui JP, Darmaun D, et al. [Childhood diet and cardiovascular risk factors]. Arch Pediatr 2010; 17(1): 51-9.
153. Costa FP, Machado SH. [Does the consumption of salt and food rich in sodium influence blood pressure in infants?]. Cien Saude Colet 2010; 15 Suppl 1: 1383-9.
154. Morinaga Y, Tsuchihashi T, Ohta Y, Matsumura K. Salt intake in 3-year-old Japanese children. Hypertens Res 2011; 34(7): 836-9.
155. Magriplis E, Farajian P, Pounis GD, Risvas G, Panagiotakos DB, Zampelas A. High sodium intake of children through 'hidden' food sources and its association with the Mediterranean diet: the GRECO study. J Hypertens 2011; 29(6): 1069-76.
156. He FJ, Marrero NM, Macgregor GA. Salt and blood pressure in children and adolescents. J Hum Hypertens 2008; 22(1): 4-11.
157. He FJ, Marrero NM, Macgregor GA. Salt intake is related to soft drink consumption in children and adolescents: a link to obesity? Hypertension 2008; 51(3): 629-34.
158. He FJ, Macgregor GA. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. Hypertension 2006; 48(5): 861-9.
159. Jurgens G, Graudal NA. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride. Cochrane Database Syst Rev 2004; (1): CD004022.
160. Garg R, Williams GH, Hurwitz S, Brown NJ, Hopkins PN, Adler GK. Low-salt diet increases insulin resistance in healthy subjects. Metabolism 2011; 60(7): 965-8.
161. Moore LL, Singer MR, Bradlee ML, Djousse L, Proctor MH, Cupples LA, et al. Intake of fruits, vegetables, and dairy products in early childhood and subsequent blood pressure change. Epidemiology 2005; 16(1): 4-11.
162. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med 2001; 344(1): 3-10.
163. Berz JP, Singer MR, Guo X, Daniels SR, Moore LL. Use of a DASH food group score to predict excess weight gain in adolescent girls in the National Growth and Health Study. Arch Pediatr Adolesc Med 2011; 165(6): 540-6.
164. Liese AD, Bortsov A, Gunther AL, Dabelea D, Reynolds K, Standiford DA, et al. Association of DASH diet with cardiovascular risk factors in youth with diabetes mellitus: the SEARCH for Diabetes in Youth study. Circulation 2011; 123(13): 1410-7.
165. Gunther AL, Liese AD, Bell RA, Dabelea D, Lawrence JM, Rodriguez BL, et al. Association between the dietary approaches to hypertension diet and hypertension in youth with diabetes mellitus. Hypertension 2009; 53(1): 6-12.
166. Couch SC, Saelens BE, Levin L, Dart K, Falciglia G, Daniels SR. The efficacy of a clinic-based behavioral nutrition intervention emphasizing a DASH-type diet for adolescents with elevated blood pressure. J Pediatr 2008; 152(4): 494-501.
167. Kelishadi R, Razaghi EM, Gouya MM, Ardalan G, Gheiratmand R, Delavari A, et al. Association of physical activity and the metabolic syndrome in children and adolescents: CASPIAN Study. Horm Res. 2007; 67(1): 46-52.
168. Kelishadi R, Cook SR, Amra B, Adibi A. Factors associated with insulin resistance and non-alcoholic fatty liver disease among youths. Atherosclerosis 2009; 204(2): 538-43.
169. Gopinath B, Hardy LL, Teber E, Mitchell P. Association between physical activity and blood pressure in prepubertal children. Hypertens Res 2011; 34(7): 851-5.
170. Andersen LB, Riddoch C, Kriemler S, Hills A. Physical activity and cardiovascular risk factors in children. Br J Sports Med 2011; 45(11): 871-6.
171. Gopinath B, Baur LA, Hardy LL, Kifley A, Rose KA, Wong TY, et al. Relationship between a range of sedentary behaviors and blood pressure during early adolescence. J Hum Hypertens 2011.
172. Carson V, Janssen I. Volume, patterns, and types of sedentary behavior and cardio-metabolic health in children and adolescents: a cross-sectional study. BMC Public Health 2011; 11: 274.
173. Yoshinaga M, Hatake S, Tachikawa T, Shinomiya M, Miyazaki A, Takahashi H. Impact of Lifestyles of Adolescents and Their Parents on Cardiovascular Risk Factors in Adolescents. J Atheroscler Thromb 2011.
174. Kelishadi R, Hashemipour M, Sarrafzadegan N, Mohammadifard N, Alikhasy H, Beizaei M, et al. Effects of a lifestyle modification trial among phenotypically obese metabolically normal and phenotypically obese metabolically abnormal adolescents in comparison with phenotypically normal metabolically obese adolescents. Matern Child Nutr. 2010; 6(3): 275-86.
175. Sweat V, Bruzzese JM, Albert S, Pinero DJ, Fierman A, Convit A. The Banishing Obesity and Diabetes in Youth (BODY) Project: Description and Feasibility of a Program to Halt Obesity-Associated Disease Among Urban High School Students. J Community Health 2011.

176. Rush E, Reed P, McLennan S, Coppinger T, Simmons D, Graham D. A school-based obesity control programme: Project Energize. Two-year outcomes. Br J Nutr 2011; 1-7.
177. Aizawa K, Shoemaker JK, Overend TJ, Petrella RJ. Effects of lifestyle modification on central artery stiffness in metabolic syndrome subjects with pre-hypertension and/or pre-diabetes. Diabetes Res Clin Pract 2009; 83(2): 249-56.
178. Collier SR, Frechette V, Sandberg K, Schafer P, Ji H, Smulyan H, et al. Sex differences in resting hemodynamics and arterial stiffness following 4 weeks of resistance versus aerobic exercise training in individuals with pre-hypertension to stage 1 hypertension. Biol Sex Differ 2011; 2(1): 9.
179. Alpert BS. Exercise as a therapy to control hypertension in children. Int J Sports Med 2000; 21 Suppl 2: S94-S96.
180. Kelishadi R, Hashemi M, Mohammadifard N, Asgary S, Khavarian N. Association of changes in oxidative and proinflammatory states with changes in vascular function after a lifestyle modification trial among obese children. Clin Chem. 2008; 54(1): 147-53.
181. Ribeiro MM, Silva AG, Santos NS, Guazzelle I, Matos LN, Trombetta IC, et al. Diet and exercise training restore blood pressure and vasodilatory responses during physiological maneuvers in obese children. Circulation 2005; 111(15): 1915-23.